ability in size and position of the left hepatic lobe and spleen.

Suggested mechanisms of ^{67}Ga localization in inflammatory sites include ^{67}Ga protein complexes collecting about the inflammatory site and the *in vivo* labeling of granulocytic leukocytes that migrate to the inflammatory focus. A decrease in circulating granulocytes has correlated with both a diminution in intensity and a delay in onset of ^{67}Ga detection in experimental inflammation.

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Cystic Medionecrosis and Dissecting Aneurysm

CYSTIC MEDIONECROSIS has been generally accepted as the cause of dissecting aortic aneurysm following its meticulous description by Erdheim more than 40 years ago. Surprisingly, he encountered the lesion not in dissecting aneurysm, but in two cases of rupture of nonsyphilitic aneurysm of the ascending aorta.

Briefly, the lesion is characterized by accumulations of mucoid substance that gradually coalesce to form cysts that interrupt the continuity of the elastica and sometimes muscle and collagen as well. Use of the term "cystic" seems objectionable because the cysts lack a distinct lining, while the term "medionecrosis" is inappropriate since necrosis is seldom demonstrable. Recent reports suggest that cystic medionecrosis is not the most frequently encountered lesion in dissecting aneurysm, but is a consistent finding only in dissections (aneurysms) that complicate the infrequent Marfan's syndrome, and in the rare instances of aneurysm of the ascending aorta that occur in the absence of syphilis.

Cystic medionecrosis occurs principally with lesions of elastic tissue rather than muscle in the aorta. Muscle lesions are considerably more frequent than elastic lesions in dissecting aneurysm and also correlate better with such clinically associated factors as hypertension and increasing age.

It seems that the term "cystic medionecrosis" has outlived its usefulness and should be discarded in favor of a designation which specifies the defective components of the vessel wall, which may include muscle, elastic tissue, collagen or ground substance.

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BCG Infections in Cancer Patients

Bacille Calmette Guérin (BCG) vaccine is being administered to some cancer patients as one element of therapy. The vaccine is a living culture of Mycobacterium bovis that has been attenuated in virulence, but the degree of attenuation has not been completely standardized throughout the world. In immunized people who are otherwise healthy, localized infections develop that sometimes progress to lymphadenitis, occasionally with suppuration of the nodes as described by Wilson. Disseminated BCG disease is even more likely to develop in patients with cancer, as reported by Aungst and co-workers.

BCG vaccine grows easily in culture, and it can be grown from infected nodes and other tissues by the same methods that are used for growing Mycobacterium tuberculosis. In culture, it has the biochemical reactions of Mycobacterium bovis, but can be further identified as the BCG strain by its inability to produce progressive disease in guinea pigs, and by resistance to lysis by mycobacteriophage 33-D.

Pasteurization of milk and control of bovine tuberculosis have almost eradicated M. bovis as a cause of human disease in the United States, and recovery of M. bovis from a patient who has received BCG vaccine strongly suggests that the organism is the vaccine strain. Both BCG and M. bovis are almost always susceptible to isonicotinic acid hydrazide (Isoniazid®), rifampin, ethambutol, streptomycin and para-aminosalicylic acid. Therefore, mycobacterial therapy can be started before the culture has been definitively identified as BCG.

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